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18. The task-hemisphere interactions were significant for right-handed females [$F(2, 28) = 12.20$; $P < .001$], reflecting a greater left hemispheric increase in blood flow for the verbal task and a greater right hemispheric increase for the spatial task; task-hemisphere interactions were also significant for left-handed males [$F(2, 28) = 9.42$; $P < .001$], reflecting a symmetrical increase for the verbal task and a greater right hemispheric increase for the spatial task.
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Destruction of Noradrenergic Neurons in Rabbit Brainstem Elevates Plasma Vasopressin, Causing Hypertension

Abstract. *When A1 noradrenergic neurons in the caudal ventrolateral medulla of rabbits are destroyed electrolytically or by local injection of the neurotoxin kainic acid, the concentration of vasopressin in plasma increases, causing hypertension. The A1 neurons may tonically inhibit the activity of vasopressin-secreting neuroendocrine cells through a direct hypothalamic projection.*

The A1 cells are a population of norepinephrine-containing neurons in the caudal ventrolateral medulla oblongata (*1*). Destruction of the area containing these neurons by electrolytic lesioning or by local administration of the neurotoxin kainic acid produces a striking cardiovascular syndrome characterized by acute hypertension, peripheral vasoconstriction, bradycardia, and florid pulmonary edema (*2*). The A1 cells do not project to the spinal cord, as was originally supposed (*3*). Rather, their axons ascend to the hypothalamus, with a major projection to supraoptic and paraventricular nuclei (*4, 5*). In these nuclei the terminals of A1 cells are concentrated around large neuroendocrine cells, which secrete arginine vasopressin (AVP) into the circulation (*5, 6*).

These anatomical observations suggest that A1 neurons may regulate the release of AVP by supraoptic and paraventricular neurons. Furthermore, they suggest that the cardiovascular syndrome observed after lesions of the A1 area may be related to increases in circulating vasopressin, a potent vasoconstrictor (*7*). We sought to determine whether A1 lesions elevate the concentration of AVP in plasma and, if so, whether this elevated AVP contributes to the hypertension associated with the lesions.

First we determined the effects of injecting kainic acid into the A1 area on mean arterial pressure (MAP) and plasma AVP. Male New Zealand White rabbits were anesthetized with urethane (1.4 g/kg, intravenously), paralyzed with curare (2 mg/kg, intravenously), and mechanically ventilated with oxygen-enriched air. A catheter was inserted into one femoral artery for continuous monitoring of arterial pressure. Samples of arterial blood (1.5 ml) were withdrawn at specified times for measurement of plasma AVP (*8*). The volume withdrawn was immediately replaced with saline. The rabbit's head was held in a Kopf stereotaxic apparatus, the dorsal surface of the medulla was surgically exposed, and mi-

Table 1. Effects of neurotoxic (kainic acid) and neuroexcitatory (L-glutamate) agents on MAP and plasma AVP in rabbits. Each agent was injected in the amount of 1 nmole dissolved in 0.25 μl of saline. Values are means ± standard errors.

Treatment	Number of rabbits	MAP (mmHg)		AVP (pg/ml)	
		Before injection	Five minutes after injection	Before injection	Five minutes after injection
Kainic acid to A1 area	10	117 ± 6	149 ± 7*	14 ± 4	70 ± 10*
Saline to A1 area	8	106 ± 6	106 ± 5	10 ± 2	14 ± 2
L-Glutamate to A1 area	7	111 ± 6	110 ± 7	12 ± 4	13 ± 4
Kainic acid to spinal trigeminal nucleus	5	124 ± 3	125 ± 3	20 ± 7	13 ± 2
Kainic acid to A1 area after cervical spinal cord transection	5	95 ± 2	128 ± 9*	38 ± 8	142 ± 45†

*Significantly different from value measured before injection ($P < .01$, Student's t-test). †$P < .05$.

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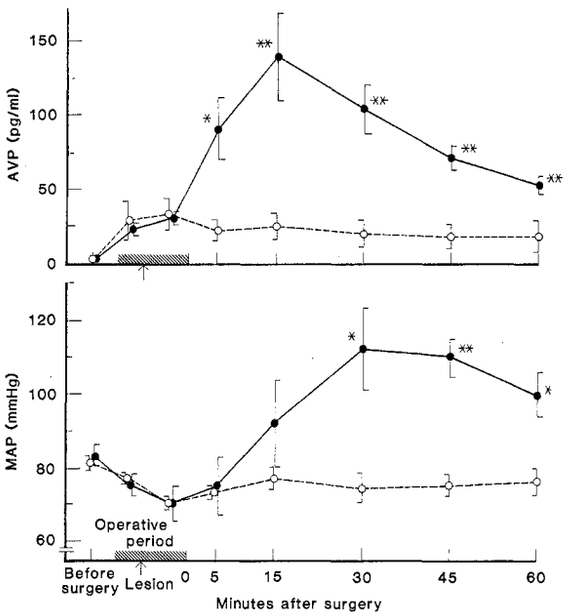

Fig. 1. Effects of electrolytic lesions of the A1 area on MAP and plasma AVP during the postoperative period after the cessation of halothane anesthesia. Each experimental value (●) is the mean ± standard error for eight rabbits, and each control value (○) is the mean ± standard error for five rabbits. Single asterisks indicate $P < .05$ and double asterisks indicate $P < .01$ (Student's t-test).

cropipettes were placed bilaterally in the A1 area (*9*).

When kainic acid (1 nmole) was injected bilaterally into the A1 area, there was a rapid reduction in MAP followed, after approximately 1 minute, by a sustained increase. This increase in MAP was associated with a fivefold increase in plasma AVP (Table 1). In contrast, neither saline injected into the A1 area nor kainic acid injected into an adjacent vasodepressor area, the spinal nucleus of the trigeminal nerve (*10*), affected MAP or AVP (Table 1).

Kainic acid has an initial depolarizing, neuroexcitatory effect followed by a delayed neurotoxic effect (*11*). To help determine which of these effects produced the observed increase in AVP, we injected the amino acid L-glutamate into the A1 area in a dose known to be neuroexcitatory but not neurotoxic (*11*). This caused a transient reduction in MAP similar to that observed with kainic acid, but no increase in MAP or AVP was observed (Table 1). This suggests that the delayed increase in MAP and the increase in AVP observed after treatment with kainic acid were due to impairment of neuronal function by this agent.

Further experiments were conducted to determine whether the elevated concentrations of AVP in plasma contributed to the hypertension. In rabbits the brain was isolated from the spinal sympathetic centers by spinal transection between the first and second cervical vertebrae. Mean arterial pressure was maintained at approximate pretransection values by an intravenous infusion of norepinephrine. In these animals injection of kainic acid into the A1 area still produced significant increases in both MAP and AVP (Table 1). Subsequent intravenous administration of an AVP antagonist (*12*) promptly and completely reversed the MAP increase.

We then sought to establish whether the cardiovascular syndrome seen in rabbits with A1 lesions (*2*) might also be related to elevated plasma AVP. Electrolytic lesions were made in the A1 area of eight rabbits anesthetized with halothane (*13*). Pre- and postoperative measures of MAP and plasma AVP were obtained from a catheter inserted into a central ear artery under local anesthesia. In five control rabbits electrodes were placed in the A1 area but no lesions were made. The results are shown in Fig. 1. After the cessation of halothane anesthesia, animals with lesions showed a sixfold increase in plasma AVP followed by a 40-mmHg increase in MAP (*14*). In control rabbits MAP was unchanged from preoperative levels and plasma AVP did not increase above the levels produced by anesthesia and surgery.

The AVP antagonist was again used to ascertain whether the elevation in MAP observed after electrolytic lesions in the A1 area was dependent on the elevated plasma AVP. Lesions were made as before in a separate group of five rabbits, and MAP increased from 79 ± 3 to 121 ± 5 mmHg after the cessation of halothane anesthesia. Blockade with AVP antagonist (10 μg/kg, intravenously) reduced MAP to 102 ± 8 mmHg within 2 minutes ($P < .01$, Student's t-test). Mean arterial pressure was not altered by the antagonist in five sham-operated rabbits.

These results demonstrate that damage to neurons in the A1 area is followed by a release of AVP into the circulation in amounts sufficient to elevate arterial pressure. In a farsighted review of the evidence available before 1974, Cross and Dyball (*15*) suggested that noradrenergic mechanisms inhibit the release of vasopressin, but they emphasized how little was known about the essential neuroanatomy. Since that time, further evidence that norepinephrine inhibits the release of AVP has been obtained (*16*), although not all studies support this conclusion (*17*). In recent neuroanatomical studies (*4, 5*), parent noradrenergic cell bodies were found in the medulla, remote from the hypothalamus and therefore amenable to experimental manipulation. The present study has taken advantage of this finding. Our results are consistent with the hypothesis that A1 noradrenergic neurons tonically inhibit the activity of vasopressin-secreting neuroendocrine cells, and demonstrate that alteration of brainstem function can alter blood pressure by releasing vasopressin.

There is increasing awareness of the physiological importance of the cardiovascular actions of vasopressin (*7*). Although the hormone has been implicated in certain forms of experimentally induced hypertension (*18*), its importance in human hypertension is controversial (*19*). However, the similarity between the syndrome in rabbits and the accelerated hypertension and "neurogenic" pulmonary edema sometimes observed in humans with brainstem damage (*20*) suggests that this clinical syndrome may have a vasopressin-dependent component.

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Localization of an Elevated Sound Source by the Green Tree Frog

Abstract. *Female green tree frogs readily localized an elevated sound source. Prior to hopping on sticks that formed a three-dimensional grid, females usually scanned laterally with their heads elevated after first scanning in the normal, nonelevated fashion. Unlike mammals and owls, frogs lack external anatomical features specialized for resolving vertical and horizontal components of source direction.*

Although acoustic orientation has been studied in many animals, the vast majority of this research has considered only the problem of sound source localization in the horizontal plane. The ability of animals to resolve the elevation of a sound source has been established only in owls, bats, monkeys, and humans (*1*).

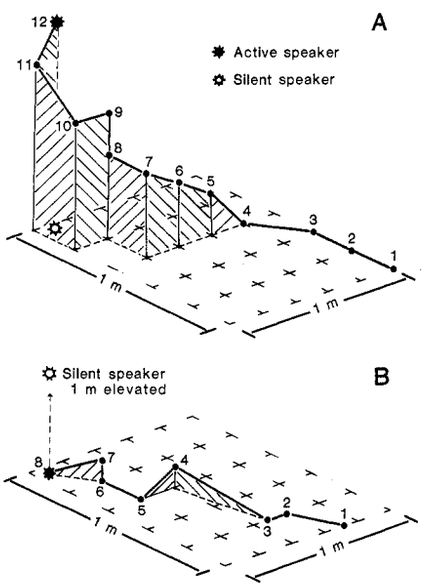

Fig. 1. Diagrams of the grid over which female tree frogs move during approaches to a speaker broadcasting synthetic mating calls. To provide for vertical movements of the animals, thin aluminum stakes (diameter, 10 mm; height, 1 m) were arranged on the grid area (1 m by 1 m); each stake position is indicated by a fine cross or other junction of fine lines in the figure. The grid of vertical 1-m stakes was stabilized by a series of crossbars at 25, 50, and 75 cm. These crossbars also served as reference points for estimating the vertical positions of the animals. Thus a spatial arrangement of many possible positions within 1 m^3 was provided. The speaker suspension plane (vertical) is indicated by the dashed line. The speaker and its support system were physically isolated from the grid so that there were no vibrational cues. (A) Diagram of a typical approach when the elevated speaker was active. The course of the frog is indicated by the heavy line, the numbers representing the frog's positions (1 to 12). The lengths of vertical lines below a number indicate the elevations to which the frog jumped or climbed at each position. (B) Diagram of a typical approach when the ground-level speaker was active, the numbers representing the frog's positions (1 to 8).

This is surprising since so many species with elaborate acoustic communication systems are arboreal or semiarboreal. Not only is sound localization in the vertical plane important for social interactions, but also it may be crucial for prey location and predator avoidance. The detection of source elevation is thought to depend on special anatomical features: pinnae in mammals and the asymmetrical (vertical) placement of the ears in owls. In this report we demonstrate that green tree frogs (*Hyla cinerea*), which lack external anatomical features specialized for acoustic orientation, readily localize an elevated sound source.

Gravid female tree frogs were collected near Savannah, Georgia, and tested within 24 hours. A three-dimensional grid of small aluminum sticks (Fig. 1) was constructed outdoors at the Oatland Island Educational Center. We suspended two identical speakers (Arso 2-inch) immediately behind the grid and isolated from contact with the grid and aluminum sticks. One speaker was about 5 cm above the ground and the second speaker about 1 m above the ground, directly over the other speaker. We broadcast synthetic mating calls [0.9 + 2.7 + 3.0 kHz (*2*)] from one or the other of the speakers, and we released females individually from a point on the ground about 1 m from the side of the grid opposite the speaker suspension plane. Experiments took place at night, and we used a dim flashlight to observe the positions and orientation movements of the frogs during phonotactic approaches to within 15 cm of a speaker.

Even before starting its approach, a frog typically made lateral scanning movements with its jaw parallel to the ground or slightly elevated. These scanning movements were observed throughout an animal's phonotactic approach to the speaker (*2*). Especially when the active speaker was elevated, the frog often repeated these lateral scanning movements after lifting its head. During a few approaches, an animal elevated and lowered its head without lateral scanning. These movements usually occurred when the animal was perched on

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